STATEMENT OF DR. GEORGE D. THURSTON, Sc. D.

TO THE

COMMITTEE ON COMMERCE

OF THE U.S. HOUSE OF REPRESENTATIVES

RE: THE HUMAN HEALTH EFFECTS OF AMBIENT OZONE EXPOSURES

MAY 8, 1997

I am George D. Thurston, a tenured Associate Professor of Environmental Medicine at the New York University (NYU) School of Medicine. My scientific research involves investigations of the human health effects of air pollution.

I am also the Director of the National Institute of Environmental Health Sciences' (NIEHS) Community Outreach and Education Program at NYU. A goal of this program is to provide an impartial scientific resource on environmental health issues to decision-makers, and that is my purpose in testifying to you here today.

Ozone (O_3) is a highly irritant gas which is formed in our atmosphere in the presence of sunlight from other "precursor" air pollutants, including nitrogen oxides and hydrocarbons. These precursor pollutants, which cause the formation of ozone, are emitted by pollution sources including automobiles, electric power plants, and industry.

Particulate Matter (PM) air pollution is composed of two major components: primary particles, or "soot", emitted directly into the atmosphere by pollution sources such as industry, electric power plants, diesel buses, and automobiles, and; "secondary particles" formed in the atmosphere from sulfur dioxide and nitrogen oxide gases, emitted by many combustion sources, including coal-burning electric power plants.

The adverse health consequences of breathing ozone or particulate matter (PM) at levels below the current U.S. National Ambient Air Quality Standards (NAAQS) are serious and well documented. This documentation includes impacts demonstrated by controlled chamber exposures and by observational epidemiology showing consistent associations between each of these pollutants and adverse impacts across a wide range of human health outcomes.

Clearly, the new EPA proposed air quality standards are based on sound science.

Observational epidemiology studies have shown compelling and consistent evidence of adverse effects by ozone and PM below the current U.S. standards. These studies statistically evaluate changes in the incidence of adverse health effects in a single population as it undergoes varying real-life exposures to pollution over time, or across multiple populations experiencing different exposures from one place to another. They are of two types: 1) population-based studies, in which aggregated counts of effects (e.g., hospital admissions counts) from an entire city might be considered in the analysis; and, 2) cohort studies, in which selected individuals, such as a group of asthmatics, are considered. Both of these types of epidemiologic studies have shown confirmatory associations between ozone and PM air pollution exposures and increased adverse health impacts, including:

- decreased lung function (a measure of our ability to breathe freely);
- more frequent respiratory symptoms;
- increased numbers of asthma attacks;

- more frequent emergency department visits;
- additional hospital admissions, and;
- increased numbers of daily deaths.

In my own research, I have found that both ozone and particulate matter air pollution are associated with increased numbers of respiratory hospital admissions in New York City, Buffalo, NY, and Toronto, Ontario, even at levels below the current standards. My results have been confirmed by other researchers considering locales elsewhere in the world. The U.S. EPA used my New York City asthma results in their "Staff Paper" when estimating the health benefits of tightening the ozone standard. However, they failed to consider other respiratory admissions affected, such as for pneumonia or bronchitis. Thus, considering the published results from various cities, the EPA analysis underpredicts the respiratory hospital admission benefits of their proposed ozone regulations by about a factor of two.

But the effects of ozone on hospital admissions are only the “tip of the iceberg” of adverse effects associated with this pollutant, and they are best viewed as indicators of the much broader spectrum of adverse health effects being experienced by the public today as a result of air pollution exposures. Most of these adverse effects are not directly recorded, however, as no central records are kept of these related, but more numerous, adverse pollutant impacts, such as increased restricted activity days and doctors visits.

In previous Congressional hearings, much discussion has centered on the hospital admissions effects of ozone, but other impacts have been largely ignored. In order to give some insight into the much larger numbers of other effects lurking beneath the surface of the ozone hospital admissions effects noted by George Wolff in Table VI-2(revised) of his November 30, 1995 closure letter to Carol Browner, I have made working estimates of the other documented adverse impacts of ozone exposure that will also be reduced in New York City, if the proposed new ozone standard were to be implemented as proposed.

The results of my analysis are presented in the figure below, entitled the "Pyramid of Annual New York City Adverse Impacts of Ozone Avoided by the Implementation of the Proposed New Standard (vs. "As Is"). This pyramid is intended to be illustrative of the enormous gaps in the table presented by Dr. Wolff, and is not presented as a peer-reviewed comprehensive documentation of all the benefits which would be accrued by achieving the EPA's proposed new standard. Please note that the figure could not be drawn "to scale". If it were drawn "to scale", the New York City (NYC) asthma admissions triangle would not even be visible, since it accounts for only approximately 0.01% of the total number of ozone related impacts noted for NYC. However, despite the fact that it visually overstates the relative size of the NYC hospital asthma admissions, and the fact that still other ozone effects cannot be considered in these calculations due to a lack of data, this figure still

makes very clear that the New York City asthma admissions counts considered in the Wolff table represent only a small fraction (far less than 1 percent) of the adverse effects of air pollution which will be avoided through the implementation of the new standard being proposed by the EPA.

The starting point of the analysis I used to estimate the "pyramid" of effects noted in the attached figure is the 265 New York City asthma admissions estimated to be avoided as a result of the implementation of the new standard, as per the top line of the Wolff chart (i.e., 385-120 = 265 admissions). First, as I noted above, there are also non-asthma respiratory admissions effects. Based upon the average ozone impacts derived from my ozone-admissions regression results for New York City and Buffalo, this indicates that the non-asthma respiratory admissions avoided (for causes such as pneumonia and bronchitis) are about 90 percent of the size of the asthma admissions, or 240/yr. Now, based on the fact that New York City hospital records indicate that 12.6% of pediatric asthma emergency department (ED) visits result in an asthma hospital admission (Barton et al, 1993), it is estimated that the ED visits associated with the 505 ozone-related respiratory admissions would amount to approximately 3,500 ozone-induced ED visits (i.e., 505 x 1/.126). Furthermore, using the ozone adverse health effect coefficients derived from the published literature by the Empire State

Pyramid of New York City, NY Annual Adverse Ozone Impacts Avoided By The Implementation of The Proposed New Standard (vs. "As Is")*

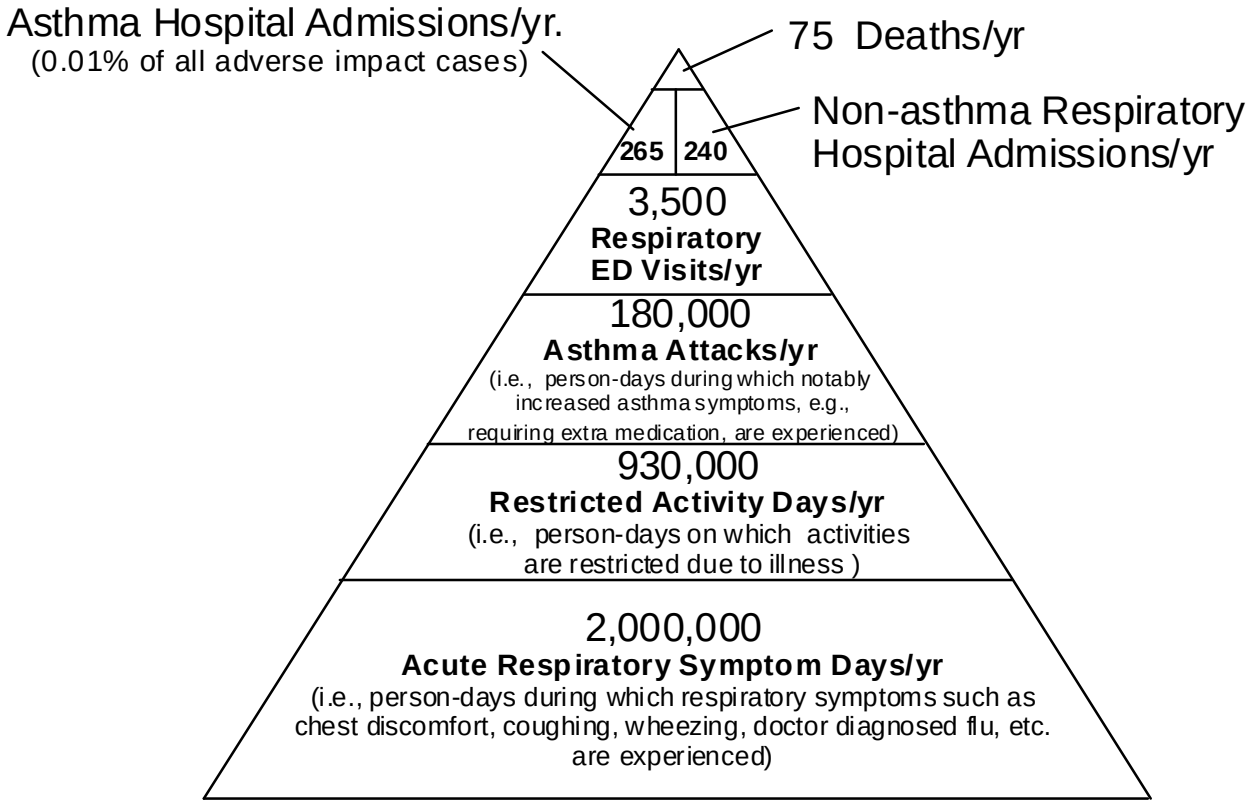


*Figure section sizes not drawn to scale.

Electric Energy Research Corporation (ESEERCO) in the New York State Environmental Externalities Cost Study (Oceana Publications, Inc., December, 1995), and ratioing the ozone effect coefficients provided in that report with that for asthma hospital admissions in New York City (used to get the 265 admissions), effects for other outcomes were derived, based on the original 265 NYC hospital admissions/day estimate. In this way, estimated annual effects to be avoided in New York City each year were also derived for:

- acute (i.e., daily) mortality,
- asthma attacks,
- restricted activity days (i.e., the total number of person-days during which some normal activities were curtailed), and
- acute respiratory symptom days (i.e., the total number of person-days during which additional respiratory symptoms would be experienced).

Some may quarrel with the specific coefficients chosen here to model the other effects, but the point remains that these other effects collectively represent large multiples of the hospital

admissions benefits noted for New York City in the chart presented at the hearing. Moreover, the categories of effects considered in the attached figure are not exhaustive by any means, but they still serve to show that the table presented by Dr. Wolff grossly underestimates the number of adverse health events that can be avoided by the meeting the proposed standard.

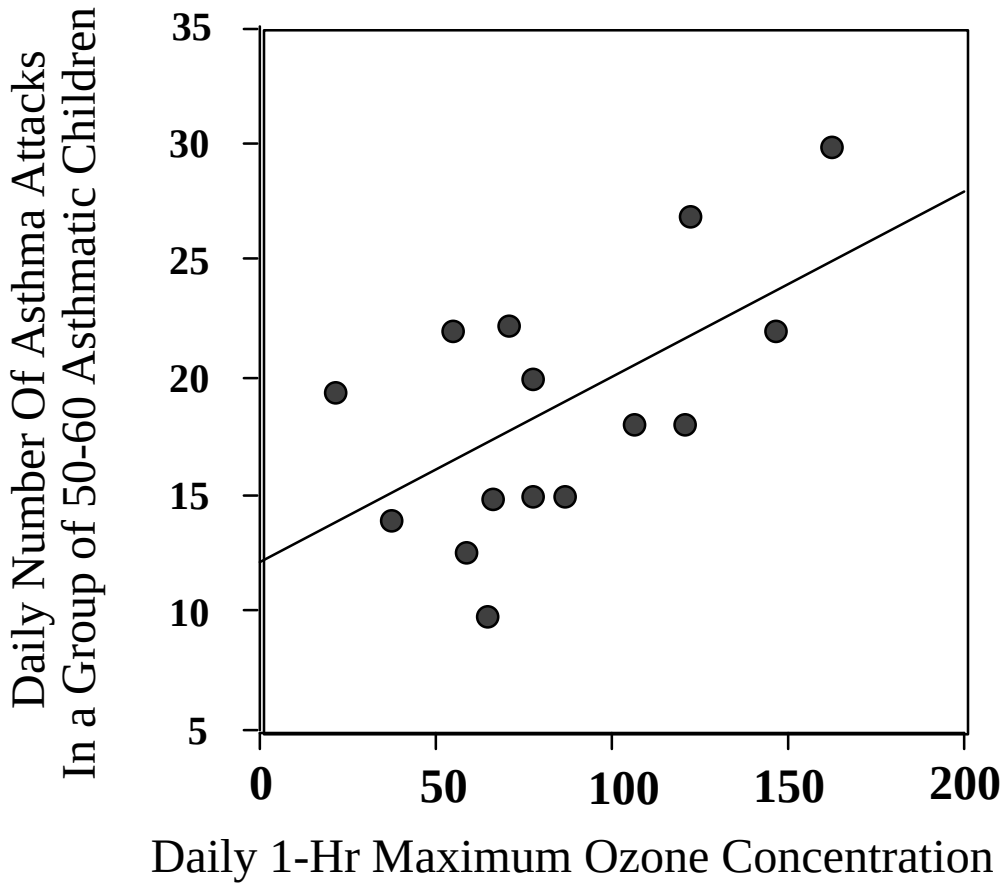
Note that the numbers in this figure have been corrected to avoid double counting of adverse health "events". For example, the number of hospital admissions has been subtracted from the total number emergency department visits, assuming that the patients would have first passed through the ED before being admitted.

Note also that this figure can be used to consider other cases in Dr. Wolff's chart as well, since all estimates have been scaled to the asthma admissions number. For example, for the difference between the existing and the proposed new standard cases, the numbers in this figure would all be divided by three (= (210-120)/(385-120) = 90/265). However, this calculation underestimates the benefits of the new standard, since it fails to account for the more rapid progress which will no doubt be able to be achieved in New York City under the new standard, when upwind counties clean up. The comparison to the "as is" case contained in the attached figure is the more apt comparison.

Finally, while there are about 7 million persons in New York City, there are a total of some 122 million persons throughout the U.S. who now live in areas exceeding the proposed O_3 standard, and will therefore also be benefited by that new standard. Thus, the New York City hospital admissions effects are best viewed as an indicator of a much broader spectrum of potentially avoidable adverse health effects being experienced by the public today as a result of air pollution exposures.

Among the important adverse effects noted above as also occurring in the New York region as a result of ozone exposure are asthma attacks. In February, the results of a study I conducted on the effects of air pollution on children at a summer "asthma" camp in Connecticut were published. This study of a group of about 55 moderate to severely asthmatic children shows that these children experience diminished lung function, increased asthma symptoms, and increased use of unscheduled asthma medications as ozone pollution levels rise. As shown in the figure below, the risk of a child having an asthma attack was found to be approximately 40 percent higher on the highest ozone days than on an average study day, with these adverse effects extending to below 120 ppb O_3.

DAILY ASTHMA ATTACKS IN CHILDREN INCREASE AS OZONE LEVELS RISE



Airway inflammation induced by ozone and PM is especially a problem for children and adults with asthma, as it makes them more susceptible to having asthma attacks, consistent with my asthma camp results. For example, recent controlled human studies (e.g., Molfino et al., 1991) have indicated that prior exposure to ozone enhances the reactivity of asthmatics to aeroallergens, such as pollens, which can trigger asthma attacks. In addition, the increased inflammation and diminished immune system ozone effects in the lung can make the elderly more susceptible to pneumonia, a major cause of illness and death in this age group.

The O_3 - morbidity associations indicated by epidemiologic studies are supported by a large body of data from controlled exposure studies that give consistent and/or supportive results, and that have demonstrated pathways by which ozone can damage the human body when it is breathed. Clinical studies have demonstrated decreases in lung function, increased frequencies of respiratory symptoms, heightened airway hyper-responsiveness, and cellular and biochemical evidence of lung inflammation in healthy exercising adults exposed to ozone concentrations at the present standard, and

at exposures as low as 80 parts per billion for 6.6 hours (e.g., Follinsbee et al., 1988, and Devlin et al, 1991).

Similarly, animal exposures to combustion-related fine particles (PM2.5) have also been shown by controlled exposure studies to have significant adverse effects on the lung, including diminished respiratory defense mechanisms, opening the lung to illness from other causes. In addition, repeated exposures to acidic fine particulate matter, a portion of the fine PM2.5 which the EPA now aims to focus on in the newly proposed reductions in the PM standard, has been shown to affect clearance in the lung in a manner similar to that of tobacco smoking, suggesting that these fine particles may have analogous long-term exposure effects on the development of Chronic Obstructive Pulmonary Disease.

The epidemiologic evidence indicating an association between PM and increased mortality and morbidity has been well documented by numerous investigators in the published literature (e.g., see Schwartz, 1997). The fact that these effects have both been shown so consistently across outcomes and from place to place is supportive of the interpretation of these associations as causal, and not due to some unknown confounder. Furthermore, controlled human and animal exposures of combustion aerosols have shown significant adverse effects by these fine particles, which are the class of particles which EPA now appropriately aims to regulate.

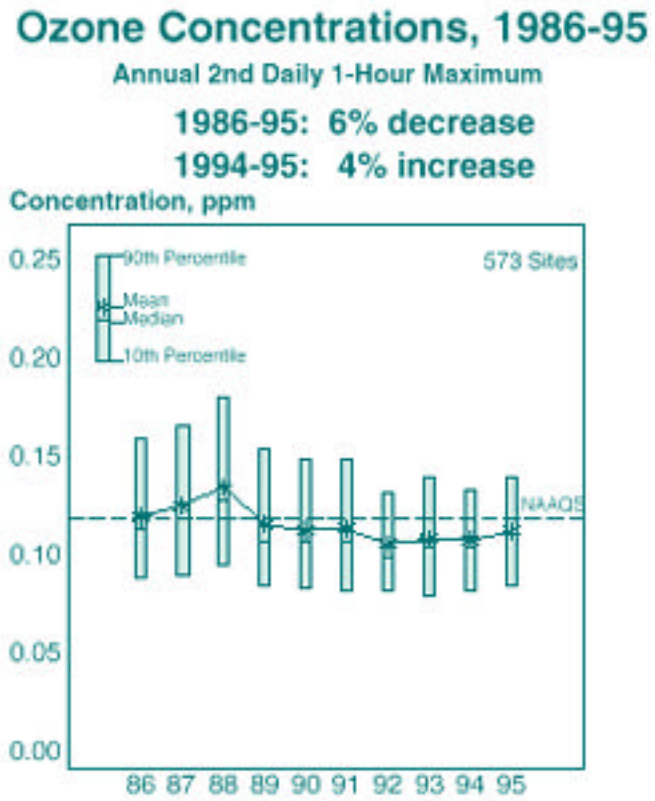
While the exact causal mechanism (i.e., the "smoking gun") of the PM-mortality association is not yet known at this time, there are biologically plausible mechanisms that are known which could account for the associations. For example, PM stresses on the lung (e.g., by inducing edema), places extra burden on the heart, which could induce fatal complications for persons with cardiac problems. Recent animal experiments by Godleski and coworkers (1996) at Harvard University confirm that exposures to elevated concentrations of ambient PM can result in cardiac related death in animals. Thus, the situation with PM and mortality is similar to many public health risks in the past, such as cholera in London a hundred years ago, or smoking over the past few decades: the epidemiology shows biologically plausible effects, but the exact mechanism by which the documented adverse impacts are effected is not yet known. This uncertainty about the exact mechanism of effect did not stop us from taking societal action against epidemiologically documented health threats, such as smoking, in the past, and should not be a deterrent to controlling the adverse consequences of particulate matter air pollution now.

Epidemiological evidence has also accumulated over recent years indicating a role by O_3 in daily human mortality, a factor not fully considered by the U.S. EPA in the latest O_3 Staff Paper or in the EPA's recent Regulatory Impact Assessment (RIA) for ozone. For example, Verhoeff, et al. (1996) used Poisson regression analysis to analyze associations between daily mortality and air pollution concentrations in Amsterdam, The Netherlands during 1986-1992, finding a daily mortality RR=1.10 per 100 ppb 1-hr daily maximum O_3, even after controlling for weather and co-pollutants. Anderson et al. (1996) investigated whether outdoor air pollution levels in London, England

influenced daily mortality during 1987-1992, finding a daily mortality RR=1.10 per 100 ppb 8-hr O_3 (RR=1.08 per 100 ppb 1-hr O_3), even after controlling for weather and co-pollutants. Samet et al. (1997) considered total daily mortality and environmental data for Philadelphia during 1973-1980, finding that, when pairs of pollutants were considered simultaneously, only the ozone coefficient consistently remained unchanged and statistically significant, with a total mortality RR of 1.02 for a 20 ppb increase in 24-hr daily average ozone. Cause of death-specific regressions indicated the largest ozone RR for respiratory deaths, consistent with biological plausibility. More recently, I have found that daily mortality also rises after high ozone days in the U.S. cities of New York City, Atlanta, Detroit, Chicago, St. Louis, Minneapolis, San Francisco, Los Angeles, and Houston, even after accounting for other factors such as season and weather, and at ozone levels below the current NAAQS standard (Thurston, 1997). I find that the overall population's risk of death rises by about 6 percent on ozone days having a 1-hour maximum of ozone that is 100 ppb above the average. The many new studies released since the EPA Criteria Document was prepared which document associations between ozone air pollution and human mortality are listed in the attached Table 1.

Once the weight of this new evidence of biologically plausible associations between acute ozone exposures and increased daily human mortality is considered, it is clearer than ever that important reductions in public health risks can be achieved by going from the present standard (equivalent to about a 90 ppb 8-hour standard) down to an 80 ppb 8-hour O_3 standard, as is being proposed by the EPA.

It has been argued that we should leave the Clean Air Act alone because rapid progress is already being made, but this is not the case for ozone. As shown in the figure below, progress regarding ambient ozone levels in the U.S. has slowed in recent years under the existing regulations.



Source: U.S. EPA, National Air Quality and Emissions Trends Report, 1995. EPA 454/R-96-005, October, 1996. RTP, NC.

Thus, it is important for committee members to realize that the downside to not acting to control these pollutants at this time is the risk that these pollutants' adverse effects will continue to occur unabated. This will result in the public unnecessarily continuing to bear the ongoing diminished quality of life and the health care costs we presently pay because of the adverse health effects of these air pollutants.

In conclusion, I would like to reiterate the key messages contained in the letter that I and 26 other air pollution researchers and physicians sent to President Clinton earlier this year:

- Please listen to the medical and scientific community on this issue.
- Exposures to O_3 and PM air pollution have been linked to medically significant adverse health effects.
- The current NAAQS for these pollutants are not sufficiently protective of public health.

Thank you for the opportunity to testify on this important issue.

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Table 1. Recent Studies Linking Ozone With Daily Mortality Not Considered by the U.S. EPA O_3 Criteria Document or Staff Paper

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Table 1. Recent Studies Linking Ozone With Daily Mortality Not Considered by the U.S. EPA O_3 Criteria Document or Staff Paper (Continued)

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